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RESTORING NITROGEN BALANCE AFTER BURN INJURY

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Introduction

Burn injury invokes a number of complex metabolic alterations that are related to subsequent morbidity and mortality. The metabolic response that ensues is characterized by extensive weight loss and negative nitrogen balance. The magnitude of the hypercatabolism is related to the extent and severity of the injury. Failure to provide the increased energy and protein requirements results in impaired wound healing, cellular dysfunction, and decreased resistance to infection.

Nutritional therapy should therefore be instituted early postburn in order to maintain host defense mechanisms and lean body mass. During the past decade, new modalities of nutritional support have aided in the achievement of positive calorie and nitrogen balance resulting in reduced postburn morbidity.

Physiology of Hypermetabolism

Hemodynamic Changes

Metabolism after burn injury is characterized by a biphasic neurohumoral response mediated through the hypothalamic-pituitary axis. The shock or "ebb" phase is short, lasting three to five days postinjury, and hypovolemia is the main focus of attention. Metabolic changes during this phase are characterized by hypotension, decreased intravascular volume, decreased blood flow, poor tissue perfusion, and hemodynamic instability.[1] Although the shift of intravascular volume into injured tissues can account for the majority of this initial response, other mechanisms are likely to be involved since an early fall in cardiac output frequently precedes the volume deficit. Energy expenditure and oxygen consumptions during this phase are generally below normal.

Following the restoration of circulating blood volume, the patient enters the catabolic or "flow" phase of the physiologic response. The rate of metabolism increases greatly, reaching maximum levels between the fifth and twelfth postburn day. This response is characterized by abnormal substrate utilization, increased heat elimination, and weight loss. The magnitude of postburn hypermetabolism is proportional to the extent of the injury. It increases linearly as burn size approaches 50% body surface area (BSA), at which point a plateau is reached at approximately two to two and one-half times that in resting, uninjured individuals. The metabolic rate may be accentu-

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ated by intervening infection[2] if there is physiologic reserve, however, it does not increase further in those patients with burns in excess of 50% BSA.

The increased metabolic expenditure remains relatively constant until the burn wound spontaneously has re-epithelialized or has been closed by skin grafting. Wound closure is not necessarily accompanied by an immediate return to normal metabolism, and energy and protein requirements may remain elevated until the wound has undergone maturation.

Hormonal Response

After burn injury, the hypothalamus receives afferent signals due to painful stimuli, hypoxia, and hypotension. Other hypothalamic stimuli released locally may include prostaglandins, interleukin-1, complement, and endotoxins. Antidiuretic hormone, growth hormone, adrenocorticotropic hormone, beta-endorphins, and possibly thyroid-stimulating hormone are initially released. The corticoids, in addition to their steroid effects, inhibit protein synthesis, facilitate amino acid mobilization from skeletal muscle, suppress insulin secretion, and liberate glucagon. During this period, alpha-adrenergic receptors are stimulated, resulting in a further inhibition of insulin release. It is generally believed, however, that these hormonal changes are not the principal factors in the initiation or maintenance of protein catabolism after injury.[3]

The second, or catabolic, phase is mediated by a complex interaction of various hormones. The catecholamines appear to be of central importance in this response and their secretion has a curvilinear correlation with burn size and metabolic rate. Pharmacologic blockade of beta-receptors substantially diminishes postburn hypermetabolism; however, complete beta-receptor blockade does not completely prevent hypermetabolism, suggesting that other mechanisms are involved in this process. The increased secretion of epinephrine and increased beta-adrenergic activity stimulates the secretion of glucagon relative to the rate of insulin. This increase in the glucagon: insulin ratio, coupled with a decrease in tissue insulin effect, is the stimulus for glycogenolysis and gluconeogenesis.

Thermoregulatory Changes

There is an increase in heat production and an elevation in core and skin temperature with the onset of postburn hypermetabolism.[4] Burn patients exhibit an increase in core temperature to 38°-39°C for maximal comfort and minimal energy expenditure. Since thermal injury destroys the water vapor barrier of the skin, evaporative water loss from the burn wound has been implicated in the etiology of the hypermetabolic response.[5] It has been suggested that surface cooling secondary to increased evaporative water loss from the burn wound may stimulate metabolic heat production in order to maintain normal body temperature. Zawacki and associates[6] blocked evaporative water loss by covering the burn wound with a water impermeable membrane under environmentally controlled conditions and found only a modest reduction in the metabolic rate. In contrast, Caldwell[7] and Barr[8] and their coworkers showed that covering the burn wound and treating the patient in a warm environment significantly reduced both the evaporative water loss and the metabolic rate.

Wilmore and colleagues[9] defined the relationship between surface cooling and hypermetabolism using a controlled ambient environment. They showed that evaporative water loss with resultant surface cooling was not the primary stimulator of hypermetabolism in the burn patient, but that an endogenous resetting of central temperature regulation was responsible for the increased energy production.[9] The burn patient, therefore, seems to be internally warm and not externally cold. Attempts to mitigate this response by increasing ambient temperatures have not resulted in a significant decrease in metabolic rate. Conversely, inadvertent cooling has been associated with a substantial increase in metabolic rate in patients with moderate injuries.

Intermediate Metabolism

During the period of hypermetabolic stress, all endogenous sources are mobilized to meet in-

creased energy demands. Carbohydrate stores such as glycogen and extracellular glucose are exhausted in less than 24 hours. In the absence of exogenous glucose, the burn patient must depend on hepatic glucose production by gluconeogenic compounds mobilized from skeletal muscle. Fat oxidation also becomes a major energy source. Burn mediated increases in catecholamines and glucagon potentiate lipase activity and increase lipolysis; however fatty acids must first be converted to an active intermediate before they can be metabolized by various enzymes. It is unclear whether lipolysis is beneficial to the burn patient because burn tissues preferentially utilize glucose.

Protein Metabolism

During stress, visceral and skeletal muscle proteins become increasingly important energy sources. Amino acids are released from structural and functional protein and are transported to the liver, chiefly in the form of alanine and glutamine, for gluconeogenesis. Alanine release from the extremities of burn patients is nearly three times that seen in normal individuals.[10] The magnitude of this release is correlated with burn size and is consistent with the accelerated gluconeogenesis and ureagenesis seen in these patients.[11]

Normal individuals endogenously produce approximately 200 grams of glucose per day, whereas noninfected burn patients produce 300-400 grams per day.[12] Owen and associates[13] have suggested that one gram of nitrogen from catabolized protein yields 3.43 grams of glucose. Since hypermetabolic burn patients excrete 20-30 grams of nitrogen per day, about 80 grams of glucose per day (20-25% of the total daily endogenous glucose production) is derived from amino acids made available by the breakdown of muscle protein. The amino groups generated as a product of the deamination process are then converted to urea which is excreted through the urine.

Nutritional Assessment

A thorough nutritional evaluation is essential for the planning and monitoring of any nutrition support regimen in burns. The assessment of caloric needs is one of the first and most important aspects of nutritional status to be considered. Fairly accurate determination of the extent of hypermetabolism is necessary because both underfeeding and overfeeding of burn patients have been associated with serious complications. Assessment should include the measurement of visceral and urinary proteins, immunologic, macronutrient and micronutrient status, and body composition. The patient's tolerance of the nutritional support regimen and daily nutrient intake should be recorded, including the number of calories provided and the proportion of those calories contributed by protein. Clinical, laboratory, and intake parameters should be monitored until wound closure has occurred.

Body Weight

It is important to monitor body weight throughout the postburn course in order to determine the adequacy of nutritional support; however, interpreting weight changes over time is somewhat difficult due to a number of confounding factors. For example, the body weight of a burn patient may increase by 20% or more with resuscitation in the first 24 hours. Subsequent shifts in fluid balance, escharotomies, amputations, bulky dressings and other attached supportive materials may cause significant errors in the determination of a true body weight[14] and must therefore be accounted for in the assessment. Weight loss, if unabated by vigorous nutritional support, is related to the percent of total body surface area burn and can be lethal.

Nitrogen Balance

Precise calorie and protein requirements are often difficult to establish in burn patients; however, accurate nitrogen balance calculations, with proper interpretation, can be used to judge the adequacy of nutritional support. The goal for nitrogen balance during repair is (+) 4 to (+) 6 grams daily. The following formula has been utilized as a means of estimating the degree of anabolism or catabolism: Nitrogen intake - (24 hr urine urea nitrogen + 4 gm). The four gram factor is added to the urine urea nitrogen to account for

nonurinary losses of nitrogen, e.g., stool and integumental nitrogen output. Additional sources of nitrogen loss include that from nasogastric suction, severe diarrhea, and exudation from the burn wound. Although these factors limit the interpretation of nitrogen balance data, this modality remains a simple, inexpensive and relatively accurate measure of protein catabolism and adequacy of nutrition support compared to body weight.

Nitrogen Loss Through the Wound

In addition to urinary nitrogen excretion, there is a significant exudative protein loss from the burn wound. This loss is difficult to measure and does not necessarily reflect changes in internal nitrogen metabolism. In the first few days after the burn, the wound exudate can contribute up to 20-25% of the total nitrogen loss.[15] Soroff and coworkers[16] demonstrated that approximately 0.2 grams of nitrogen per percent burn per day were lost through burn exudate during the first week after injury, with declining losses thereafter. In his study of nitrogen loss in burn patients, Soroff categorized four periods following the first week after injury: catabolism, anabolism, postgrafting anabolism, and convalescence. He showed that the rate of utilization of nitrogen did not differ among the four periods but that the obligatory nitrogen loss and the requirements for nitrogen equilibrium decreased as the postburn course progressed through convalescence. Consequently, it would appear that exudative protein loss might be effectively reduced by early excision and grafting of the burn wound.

Urine Urea Nitrogen

Urine urea nitrogen (UUN) accounts for 85-90% of the total urinary nitrogen loss and is markedly increased during the catabolic phase.[15] It is a unique variable because it is not affected by nitrogen loss from the wound or other sources. Measurement of the UUN level is, in effect, a measurement of internal nitrogen metabolism and can be performed easily on a 24-hour urine collection.

The increased UUN level is a result of accelerated skeletal muscle proteolysis and is a direct reflection of the magnitude of injury. Urea is synthesized in ureotelic organisms as the major vehicle for the excretion of surplus nitrogen. O'Keefe and associates[17] have attributed the surplus nitrogen to a relative failure of protein synthesis rather than an enhanced rate of breakdown of pre-existing protein. More recently, Kien and coworkers[4] studied the rate of whole body protein synthesis and breakdown in burned children. They showed that the rates of both protein synthesis and breakdown were increased in patients with large burns. They also proposed that whole body protein turnover is partly responsible for postburn hypermetabolism because of the high energy cost of protein synthesis.

Nutritional Requirements

Calories

Energy requirements may be estimated or measured. Obviously, measured data provides a better indicator of actual needs. With the increased availability of portable metabolic carts, bedside determination of energy expenditure from oxygen consumption and carbon dioxide production is frequently employed at many burn centers.[18] An additional 20-30% should be added to resting energy expenditure measurements to account for energy elevations that occur with physical therapy, dressing changes and other events that heighten needs. Ideally, indirect calorimetry should be performed at regular intervals in order to tailor the alimentation program to the patient's changing condition.

As an alternative to indirect calorimetry, mathematical derivations have been constructed to provide estimates of energy requirements based on factors such as percent burn, weight, height, body surface area, age, sex and activity. There are at least 25 formulas that can be used to predict the energy needs of burn patients. Several of the more widely used formulas are listed in the Table. The Curreri formula[19] has enjoyed popularity for its simplicity and applicability. It uses the initial percent burn and the preburn body weight to predict energy needs. The Harris-Benedict

equation has been adapted by Long[20] for burn injury by multiplying the equation by a factor to account for the increase in energy expenditure due to burns. Although this formula is widely used in estimating energy expenditure postburn, none of the formulas provide for the great variation observed among individual patients. Variables influencing energy expenditure, including body composition, age, sex, preburn nutritional status, extent of injury, healing, room temperature, pain and fever are often lacking in caloric equations. Effectors of body weight such as edema, amputations and dressings introduce other potential sources for error when incorporated into mathematical calculations. Energy predicting equations have been extensively reviewed as well as evaluated for clinical validity elsewhere.[14,18,21] In short, the clinician should utilize indirect calorimetry or select one of the predictive formulas and modify therapy based upon nutritional assessment data.

Protein

Protein requirements are also increased in burn victims, because protein synthesis demands are enhanced for wound healing and significant amounts of nitrogen are lost in the exudate from the wound, excisions, loss of blood and the degradation of muscle mass. Alexander and associates[22] have shown that significant amounts of protein are required to achieve nitrogen balance and immunologic benefit. By providing 22-25% of calories as protein (a calorie to nitrogen ratio of approximately 75:1), burned children were able to achieve positive nitrogen balance as well as demonstrating marked improvements in opsonic

Table • Formulas for Calculating Energy and Protein Requirements for Burned Patients

REFERENCE	AGE	%BSAB	CALORIES PER DAY	PROTEIN PER DAY
Alexander[23]	Child	Any %		22-25% of Kcal
Curreri[19]	0-1	<50%	Basal + (15 x % burn)	
	1-3	<50%	Basal + (25 x % burn)	
	4-15	<50%	Basal + (40 x %burn)	
	16-59	Any %	(25 x W) + (40 x % burn)	3 gm/kg
	>60	Any %	Basal + (65 x % burn)	
Long's modification[20] of the Harris-Benedict equation	Male	Any %	(66.47 + 13.7 W + 5.0 H - 6.76 A) x (activity factor) x (injury factor)	
	Female	Any %	(655.1 + 9.56 W + 1.85 H - 6.68A) x (activity factor) x (injury factor)	
			Activity factor: (a) confined to bed, use 1.2 (b) out of bed, use 1.3	Injury factor: (a) severe thermal burn, use 2.0
Soroff[16]	Adult	Any %	3500/m² BSA	
Wilmore[15]	Adult	Any %	2000/m² BSA	94 gm/m² BSA

BSA = body surface area
BSAB = body surface area burned
BMR = basal metabolic rate
W = weight in kg H = height in cm A = age in years

index and serum transferrin levels, compared to patients receiving adequate calories but less protein. This study advises providing one fourth of the energy needs as protein. This recommendation and other guidelines for protein intake in burns are listed in the Table.

Clinical Aspects of Nutritional Support

The initial approach to the problem of nutritional support in the burn patient should be the control of environmental factors. Calorie requirements can be mitigated by reducing pain and emotional stress factors. The need for uninterrupted sleeping intervals is critically important. Appropriate utilization of narcotics, sedatives, and supportive psychological intervention are helpful. Occlusive dressings[7] and ambient temperatures of 28°-32°C can minimize metabolic expenditure for maintenance of core temperature. In addition, it is extremely important for the patient to exercise in order to prevent or minimize the skeletal muscle catabolism associated with prolonged immobilization. Last, prevention of infection and closure of the burn wound are of utmost importance.

Oral Intake

It is important to supply sufficient nutritional support during the postburn hypermetabolic state. In general, patients with burns less than 20% total body surface area are capable of meeting their energy and protein needs by diet alone. Careful attention should be paid to the patient's food preferences and meals may be fortified to increase calorie intake. In addition, protein supplements can be added to provide sufficient amounts of this substrate. Drastic alterations of eating patterns that require patients to consume large volumes of food are not recommended. In order to provide optimal diet therapy without contributing to obesity, enrichment of foods with hidden calories and protein offers the best solution.

Enteral Nutrition

Indications & Techniques

Whenever possible, the enteral route is preferred to utilize the absorptive mechanisms of the gastrointestinal tract. Inadequate protein or caloric intake causes catabolism of body tissues to provide energy and amino acids for the synthesis of protein. In general, patients with burns in excess of 20% total body surface area will require enteral supplementation. Alert patients with a functional gastrointestinal tract usually tolerate nasogastric tube feedings quite well. The insertion of a nasoduodenal tube under fluoroscopy to position the tube in the third portion of the duodenum allows for the provision of nutritional support during ileus. Simultaneous gastric decompression may be maintained via a nasogastric tube connected to suction. Nasogastric suction may be discontinued once gastric motility has returned.

Diligent monitoring is necessary to prevent potential complications. Continuous infusion utilizing a tube feeding pump is recommended. Once tube placement is confirmed, enteral nutrition may be begun at full-strength. The initial hourly rate should meet one-half the calculated caloric needs. Feedings are then advanced 5 ml/hr in children and 10 ml/hr in adults until individual caloric goals are achieved, thus delivering the desired energy needs without delay. Initially, feedings are delivered continuously 24 hrs/day. As the patient's condition and appetite improve, feedings can be held for several hours at mealtime to encourage oral intake. Eventually, tube feedings are delivered only at night to augment nutritional intake.

Feeding During Ileus

Gastrointestinal paralysis occurs following a major thermal injury. Traditionally, enteral nutrition has been withheld up to a week postburn pending the resolution of ileus. It is now known, however, that the small bowel maintains motility and absorptive capabilities during this period. Mochizuki et al[23] have demonstrated that immediate enteral feeding postburn prevented the hypermetabolic response observed in control animals not fed for 24-72 hours following injury. In addition, early fed animals showed less weight loss, positive nitrogen balance and a decreased incidence of diarrhea.

A prospective study designed to evaluate the

safety and efficacy of immediate nutritional support postburn demonstrated that the early fed group consistently required 18-25% fewer calories than predicted, while the control group required 0.3-5.5% more calories than predicted.[24] Trends toward improved measures of protein stores, total protein and albumin were noted in the early fed group throughout the study period suggesting improved nutritional status. The early fed group also demonstrated a decrease in the total number of infectious complications and days of systemic antibiotic therapy. No patient in either group experienced aspiration and there were no differences in the incidence of complications possibly related to nutritional support.

Intraoperative Feeding

It is common practice to maintain patients NPO prior to and during operative procedures; however, it has recently been shown that enteral feedings can be continued during the perioperative and intraoperative periods without complication.[25] Patients supported in this fashion did not develop significant caloric deficits and required less albumin supplementation to maintain adequate serum levels. Diligent monitoring for tube position, gastric reflux and potential complications allow for the safe continuation of enteral support during these periods.

Parenteral Nutrition

While enteral alimentation is the preferred route to deliver nutrients to the burn patient, under certain circumstances, intravenous feeding can become a necessary and even life-saving part of burn management. If the gastrointestinal tract is unavailable because of concurrent abdominal trauma, persistent intestinal ileus, or poor tolerance of enteral feeding, nutritional support should be provided intravenously. Frequently, the enormous caloric demands cannot be met entirely by enteral support. The development of vomiting, abdominal distention, or intractable diarrhea limit the number of calories that can be delivered by the enteral route. In these situations, the use of TPN is extremely beneficial in supplying the caloric deficit. Gastrointestinal complications associated with large burns that are definite indications for intravenous feeding include stress ulceration of the stomach or duodenum (Curling's ulcer), severe pancreatitis, septic ileus or pseudo-obstruction of the colon. Occasionally superior mesenteric artery syndrome is observed in burn patients following a period of rapid weight loss, necessitating the use of total parenteral nutrition.

Total parenteral nutrition is not a risk-free procedure and is associated with a wide variety of mechanical, metabolic, and infectious complications in burns.[26] Its use should be reserved for those patients unable to tolerate nutritional support via the enteral route. When employed, its use should be limited to as brief a period of time as possible. The central venous catheter is preferably inserted through unburned tissue using either the subclavian or internal jugular vein. Insertion and utilization of an indwelling central venous catheter imposes significant risks including pneumothorax, microbial contamination of the catheter, phlebitis, venous thrombosis, air embolism, pulmonary or hepatic dysfunction, and nutrient imbalances, to name a few. Most of these complications can be minimized or prevented by the adherence to recommended protocols, meticulous catheter care, experienced personnel, and an ongoing monitoring system. Early recognition is the key to successful management of any complication.

Summary

The metabolic response to burn injury is characterized by weight loss and marked protein wasting. This phenomenon is mediated hormonally, resulting in hypermetabolism. Energy expenditure increases linearly with the extent of burn injury, reaching a plateau of twice resting energy expenditure when 50% of the total body surface area is involved. It is therefore essential to minimize other factors that may further augment postburn catabolism. Occlusive dressings, a warm ambient environment, analgesics, and timely closure of the burn wound are all important therapeutic measures in this regard. Furthermore, it is imperative to institute early nutritional support in order to offset the negative metabolic effects of severe burn injury. ▲

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